

=> file biosis, medline, scisearch, embase
COST IN U.S. DOLLARS

SINCE FILE	TOTAL
ENTRY	SESSION
0.21	0.21

FILE 'BIOSIS' ENTERED AT 16:58:44 ON 28 MAR 2007
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=> s sildenafil and zaprinast
L1 324 SILDENAFIL AND ZAPRINAST

=> display l1 all 324

L1 ANSWER 324 OF 324 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
AN 96234676 EMBASE <<LOGINID::20070328>>
DN 1996234676
TI Sildenafil (Viagra(TM)), a potent and selective inhibitor of type 5 CGMP phosphodiesterase with utility for the treatment of male erectile dysfunction.
AU Terrett N.K.; Bell A.S.; Brown D.; Ellis P.
CS Department of Discovery Chemistry, Pfizer Central Research, Sandwich, Kent CT13 9NJ, United Kingdom
SO Bioorganic and Medicinal Chemistry Letters, (1996) Vol. 6, No. 15, pp. 1819-1824. .
ISSN: 0960-894X CODEN: BMCLE8
CY United Kingdom
DT Journal; Article
FS 028 Urology and Nephrology
029 Clinical Biochemistry
030 Pharmacology
037 Drug Literature Index
LA English
SL English
ED Entered STN: 17 Sep 1996
Last Updated on STN: 17 Sep 1996
AB 5-(2'-Alkoxyphenyl)pyrazolo[4,3-d]pyrimidin-7-ones, and in particular our preferred compound, sildenafil (VIAGRA(TM)), discovered through a rational drug design programme, are potent and selective inhibitors of the type 5 cGMP phosphodiesterase from both rabbit platelets and human corpus cavernosum. Sildenafil is currently in the clinic for the oral treatment of male erectile dysfunction.
CT Medical Descriptors:
*corpus cavernosum
*enzyme inhibition
*penis erection
*sexual dysfunction
animal cell
article
controlled study
drug design
human
human tissue
nonhuman
rabbit

structure activity relation
thrombocyte

Drug Descriptors:

*cyclic gmp phosphodiesterase: EC, endogenous compound
*phosphodiesterase inhibitor: CM, drug comparison
*phosphodiesterase inhibitor: AN, drug analysis
*phosphodiesterase inhibitor: DV, drug development
 *sildenafil: CM, drug comparison
 *sildenafil: AN, drug analysis
 *sildenafil: DV, drug development
 zaprinas: CM, drug comparison

RN (cyclic gmp phosphodiesterase) 9068-52-4; (sildenafil)
139755-83-2; (zaprinas) 37762-06-4
CN Viagra

=> FIL STNGUIDE

COST IN U.S. DOLLARS

SINCE FILE

TOTAL

ENTRY

SESSION

FULL ESTIMATED COST

8.70

8.91

FILE 'STNGUIDE' ENTERED AT 16:59:56 ON 28 MAR 2007

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FILE CONTAINS CURRENT INFORMATION.

LAST RELOADED: Mar 23, 2007 (20070323/UP).

=> display l1 all 323

YOU HAVE REQUESTED DATA FROM FILE 'BIOSIS, MEDLINE, SCISEARCH, EMBASE' - CONTINUE?

(Y)/N:y

L1 ANSWER 323 OF 324 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN

AN 97375812 EMBASE <<LOGINID::20070328>>

DN 1997375812

TI The role of phosphodiesterase inhibitors in impotence.

AU Eardley I.

CS I. Eardley, Pyrah Department of Urology, St James' University Hospital, Leeds, United Kingdom

SO Expert Opinion on Investigational Drugs, (1997) Vol. 6, No. 12, pp. 1803-1810.

Refs: 39

ISSN: 1354-3784 CODEN: EOIDER

CY United Kingdom

DT Journal; General Review

FS 028 Urology and Nephrology

030 Pharmacology

037 Drug Literature Index

LA English

SL English

ED Entered STN: 30 Dec 1997

Last Updated on STN: 30 Dec 1997

AB Penile erection is mediated by nitric oxide (NO) and its second messenger cyclic guanosine monophosphate (cGMP). The activity of cGMP is modulated by the enzyme phosphodiesterase (PDE). The most important physiological isoform of the enzyme in the penis is PDES. Sildenafil is a specific inhibitor of this enzyme which potentiates nitrenergic cavernosal relaxation in vitro and in animals. Clinical studies suggest that it is effective in the treatment of male erectile dysfunction (MED), which would make it the first orally active therapy for this condition.

CT Medical Descriptors:
 *impotence: DT, drug therapy
 human
 male
 oral drug administration
 pharmacodynamics
 review
 Drug Descriptors:
 *phosphodiesterase inhibitor: DV, drug development
 *phosphodiesterase inhibitor: DT, drug therapy
 *phosphodiesterase inhibitor: PK, pharmacokinetics
 *phosphodiesterase inhibitor: PD, pharmacology
 *sildenafil: DV, drug development
 *sildenafil: DT, drug therapy
 *sildenafil: PD, pharmacology
 cyclic gmp: EC, endogenous compound
 dipyridamole: PD, pharmacology
 phosphodiesterase: EC, endogenous compound
 zaprinast: PD, pharmacology
 RN (sildenafil) 139755-83-2; (cyclic gmp) 7665-99-8; (dipyridamole)
 58-32-2; (zaprinast) 37762-06-4

=> s "calcium blocker" or alpha-blocker or beta-blocker or "ergot alkanoid" or
 "vasoactive intestinal peptide" or "dopamine agonist" or "opioid antagonist"
 prostaglandin or "endothelin antagonist" or "potassium channel activator"

0 "CALCIUM"
 0 "BLOCKER"
 0 "CALCIUM BLOCKER"
 ("CALCIUM" (W) "BLOCKER")
 0 ALPHA
 0 BLOCKER
 0 ALPHA-BLOCKER
 (ALPHA (W) BLOCKER)
 0 BETA
 0 BLOCKER
 0 BETA-BLOCKER
 (BETA (W) BLOCKER)
 0 "ERGOT"
 0 "ALKANOID"
 0 "ERGOT ALKANOID"
 ("ERGOT" (W) "ALKANOID")
 0 "VASOACTIVE"
 0 "INTESTINAL"
 1 "PEPTIDE"
 0 "VASOACTIVE INTESTINAL PEPTIDE"
 ("VASOACTIVE" (W) "INTESTINAL" (W) "PEPTIDE")
 0 "DOPAMINE"
 0 "AGONIST"
 0 "DOPAMINE AGONIST"
 ("DOPAMINE" (W) "AGONIST")
 0 "OPIOID"
 0 "ANTAGONIST"
 0 PROSTAGLANDIN
 0 "OPIOID ANTAGONIST" PROSTAGLANDIN
 ("OPIOID" (W) "ANTAGONIST" (W) PROSTAGLANDIN)
 0 "ENDOTHELIN"
 0 "ANTAGONIST"
 0 "ENDOTHELIN ANTAGONIST"
 ("ENDOTHELIN" (W) "ANTAGONIST")
 0 "POTASSIUM"
 0 "CHANNEL"
 0 "ACTIVATOR"
 0 "POTASSIUM CHANNEL ACTIVATOR"

```

L2      ("POTASSIUM" (W) "CHANNEL" (W) "ACTIVATOR")
0 "CALCIUM BLOCKER" OR ALPHA-BLOCKER OR BETA-BLOCKER OR "ERGOT
  ALKANOID" OR "VASOACTIVE INTESTINAL PEPTIDE" OR "DOPAMINE AGONIS
  T" OR "OPIOID ANTAGONIST" PROSTAGLANDIN OR "ENDOTHELIN ANTAGONIST
  " OR "POTASSIUM CHANNEL ACTIVATOR"

```

```

=> s "calcium blocker" or alpha-blocker or beta-blocker or "ergot alkanoid" or
"vasoactive intestinal peptide" or "dopamine agonist" or "opioid antagonist" or
prostaglandin or "endothelin antagonist" or "potassium channel activator"

```

```

0 "CALCIUM"
0 "BLOCKER"
0 "CALCIUM BLOCKER"
  ("CALCIUM" (W) "BLOCKER")
0 ALPHA
0 BLOCKER
0 ALPHA-BLOCKER
  (ALPHA (W) BLOCKER)
0 BETA
0 BLOCKER
0 BETA-BLOCKER
  (BETA (W) BLOCKER)
0 "ERGOT"
0 "ALKANOID"
0 "ERGOT ALKANOID"
  ("ERGOT" (W) "ALKANOID")
0 "VASOACTIVE"
0 "INTESTINAL"
1 "PEPTIDE"
0 "VASOACTIVE INTESTINAL PEPTIDE"
  ("VASOACTIVE" (W) "INTESTINAL" (W) "PEPTIDE")
0 "DOPAMINE"
0 "AGONIST"
0 "DOPAMINE AGONIST"
  ("DOPAMINE" (W) "AGONIST")
0 "OPIOID"
0 "ANTAGONIST"
0 "OPIOID ANTAGONIST"
  ("OPIOID" (W) "ANTAGONIST")
0 PROSTAGLANDIN
0 "ENDOTHELIN"
0 "ANTAGONIST"
0 "ENDOTHELIN ANTAGONIST"
  ("ENDOTHELIN" (W) "ANTAGONIST")
0 "POTASSIUM"
0 "CHANNEL"
0 "ACTIVATOR"
0 "POTASSIUM CHANNEL ACTIVATOR"
  ("POTASSIUM" (W) "CHANNEL" (W) "ACTIVATOR")

```

```

L3      0 "CALCIUM BLOCKER" OR ALPHA-BLOCKER OR BETA-BLOCKER OR "ERGOT
  ALKANOID" OR "VASOACTIVE INTESTINAL PEPTIDE" OR "DOPAMINE AGONIS
  T" OR "OPIOID ANTAGONIST" OR PROSTAGLANDIN OR "ENDOTHELIN ANTAGON
  IST" OR "POTASSIUM CHANNEL ACTIVATOR"

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=> file biosis, medline, scisearch, embase
COST IN U.S. DOLLARS

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FULL ESTIMATED COST

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SINCE FILE      TOTAL
ENTRY          SESSION
1.08           18.75

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FILE 'BIOSIS' ENTERED AT 17:11:21 ON 28 MAR 2007
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FILE 'MEDLINE' ENTERED AT 17:11:21 ON 28 MAR 2007

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FILE 'SCISEARCH' ENTERED AT 17:11:21 ON 28 MAR 2007

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FILE 'EMBASE' ENTERED AT 17:11:21 ON 28 MAR 2007

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=> s "calcium blocker" or alpha-blocker or beta-blocker or "ergot alkanoid" or "vasoactive intestinal peptide" or "dopamine agonist" or "opioid antagonist" or prostaglandin or "endothelin antagonist" or "potassium channel activator"

L4 519080 "CALCIUM BLOCKER" OR ALPHA-BLOCKER OR BETA-BLOCKER OR "ERGOT ALKANOID" OR "VASOACTIVE INTESTINAL PEPTIDE" OR "DOPAMINE AGONIST" OR "OPIOID ANTAGONIST" OR PROSTAGLANDIN OR "ENDOTHELIN ANTAGONIST" OR "POTASSIUM CHANNEL ACTIVATOR"

=> s L4 and "chronic obstructive pulmonary"

L5 591 L4 AND "CHRONIC OBSTRUCTIVE PULMONARY"

=> display l5 all 591

L5 ANSWER 591 OF 591 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN

AN 75208458 EMBASE <<LOGINID::20070328>>

DN 1975208458

TI Personal experience with prostaglandin E2 in obstructive respiratory disease.

AU Maulide I.

CS Serv. Doencas Pulmon., Hosp. Sta Maria, Lisboa, Portugal

SO Pneumologia, (1974) Vol. 5, No. 4, pp. 241-247. .

CODEN: PNMLAB

DT Journal

FS 037 Drug Literature Index

003 Endocrinology

015 Chest Diseases, Thoracic Surgery and Tuberculosis

LA English

AB The main characteristics of the prostaglandins are described.

The results are presented of a trial in which the responses to an aerosol of PGE2 were studied in 10 normal adults and 12 patients suffering from chronic obstructive pulmonary disease. A further study was carried out in 10 of the latter patients who are treated with PGE2 aerosol for 60 days. PGE2 has a bronchodilating action, particularly in non infected bronchioli. The mean duration of this effect is from 6 to 7 hr. Infection is an important factor to consider as it considerably reduces the effect of PGE2 in these patients. The difficulty in obtaining a metered aerosol of PGE2 constitutes a serious handicap for the ambulatory use of this substance in these patients.

CT Medical Descriptors:

*aerosol

*asthma

*bronchodilatation

*chronic obstructive lung disease

*clinical study

*breathing

theoretical study

major clinical study

therapy

inhalational drug administration

normal human

Drug Descriptors:

*prostaglandin

*prostaglandin e2

RN (prostaglandin e2) 363-24-6

=> s L5 and "phosphodiesterase inhibitor"

L6 13 L5 AND "PHOSPHODIESTERASE INHIBITOR"

=> display 16 all 13

L6 ANSWER 13 OF 13 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
AN 1998154822 EMBASE <<LOGINID::20070328>>
TI New therapies for chronic obstructive pulmonary disease.
AU Barnes P.J.
CS Prof. P.J. Barnes, Department of Thoracic Medicine, National Heart and Lung Institute, Imperial College, London SW3 6LY, United Kingdom
SO Thorax, (1998) Vol. 53, No. 2, pp. 137-147. .
Refs: 139
ISSN: 0040-6376 CODEN: THORA7
CY United Kingdom
DT Journal; General Review
FS 006 Internal Medicine
015 Chest Diseases, Thoracic Surgery and Tuberculosis
030 Pharmacology
037 Drug Literature Index
LA English
ED Entered STN: 18 Jun 1998
Last Updated on STN: 18 Jun 1998
CT Medical Descriptors:
*chronic obstructive lung disease: DT, drug therapy
*chronic obstructive lung disease: TH, therapy
bronchodilatation
smoking cessation
beta adrenergic stimulation
immunomodulation
drug efficacy
drug mechanism
human
review
priority journal
Drug Descriptors:
*bronchodilating agent: DT, drug therapy
*bronchodilating agent: PD, pharmacology
*leukotriene b4 receptor antagonist: DT, drug therapy
*leukotriene b4 receptor antagonist: PD, pharmacology
*prostaglandin derivative: DT, drug therapy
*prostaglandin derivative: PD, pharmacology
*macrolide: DT, drug therapy
*macrolide: PD, pharmacology
*antioxidant: DT, drug therapy
*antioxidant: PD, pharmacology
*corticosteroid: DT, drug therapy
*corticosteroid: PD, pharmacology
misoprostol: DT, drug therapy
misoprostol: PD, pharmacology
butaprost: DT, drug therapy
butaprost: PD, pharmacology
erythromycin: DT, drug therapy
erythromycin: PD, pharmacology
clarithromycin: DT, drug therapy
clarithromycin: PD, pharmacology
roxithromycin: DT, drug therapy
roxithromycin: PD, pharmacology
1 [3 (cyclopentyloxy) 4 methoxyphenyl] 1 phenyl 2 (4 pyridyl)ethane: DT, drug therapy
1 [3 (cyclopentyloxy) 4 methoxyphenyl] 1 phenyl 2 (4 pyridyl)ethane: PD, pharmacology
immunomodulating agent: DT, drug therapy
immunomodulating agent: PD, pharmacology

muscarinic m2 receptor: EC, endogenous compound
 darifenacin: DT, drug therapy
 darifenacin: PD, pharmacology
 muscarinic m3 receptor antagonist: DT, drug therapy
 muscarinic m3 receptor antagonist: PD, pharmacology
 revatropate: DT, drug therapy
 revatropate: PD, pharmacology
 atropine: DT, drug therapy
 ipratropium bromide: DT, drug therapy
 immunoglobulin enhancer binding protein: EC, endogenous compound
 leukotriene b4: EC, endogenous compound
 interleukin 8: EC, endogenous compound
 2 [2 propyl 3 [3 [2 ethyl 4 (4 fluorophenyl) 5
 hydroxyphenoxy]propoxy]phenoxy]benzoic acid: DT, drug therapy
 2 [2 propyl 3 [3 [2 ethyl 4 (4 fluorophenyl) 5
 hydroxyphenoxy]propoxy]phenoxy]benzoic acid: PD, pharmacology
 7 [3 [2 cyclopropylmethyl 3 methoxy 4 [(methylamino)carbonyl]phenoxy]propoxy] 3,4 dihydro 8 propyl 2h 1 benzopyran 2 propanoic acid: DT, drug therapy
 7 [3 [2 cyclopropylmethyl 3 methoxy 4 [(methylamino)carbonyl]phenoxy]propoxy] 3,4 dihydro 8 propyl 2h 1 benzopyran 2 propanoic acid: PD, pharmacology
 1 [3 (4 biphenylmethyl) 4 hydroxychroman 7 yl]cyclopentanecarboxylic acid: DT, drug therapy
 1 [3 (4 biphenylmethyl) 4 hydroxychroman 7 yl]cyclopentanecarboxylic acid: PD, pharmacology
 sb 201146: DT, drug therapy
 sb 201146: PD, pharmacology
 phosphodiesterase inhibitor: DT, drug therapy
 phosphodiesterase inhibitor: PD, pharmacology
 4 cyano 4 [3 cyclopentyloxy 4 methoxy phenyl] 1 cyclohexanecarboxylic acid: DT, drug therapy
 4 cyano 4 [3 cyclopentyloxy 4 methoxy phenyl] 1 cyclohexanecarboxylic acid: PD, pharmacology
 cp 80633: DT, drug therapy
 cp 80633: PD, pharmacology
 unindexed drug
 unclassified drug
 RN (misoprostol) 59122-46-2, 59122-48-4; (butaprost) 69648-38-0;
 (erythromycin) 114-07-8, 70536-18-4; (clarithromycin) 81103-11-9;
 (roxithromycin) 80214-83-1; (darifenacin) 133099-04-4, 133099-07-7;
 (revatropate) 149926-91-0; (atropine) 51-55-8, 55-48-1; (ipratropium bromide) 22254-24-6; (leukotriene b4) 71160-24-2; (interleukin 8) 114308-91-7; (2 [2 propyl 3 [3 [2 ethyl 4 (4 fluorophenyl) 5
 hydroxyphenoxy]propoxy]phenoxy]benzoic acid) 161172-51-6; (7 [3 [2
 cyclopropylmethyl 3 methoxy 4 [(methylamino)carbonyl]phenoxy]propoxy] 3,4
 dihydro 8 propyl 2h 1 benzopyran 2 propanoic acid) 141059-52-1,
 153632-99-6, 153633-01-3, 153633-02-4; (1 [3 (4 biphenylmethyl) 4
 hydroxychroman 7 yl]cyclopentanecarboxylic acid) 158081-99-3
 CN Ly 293111; Sc 53228; Cp 105696; Sb 201146; Sb 207499; Cp 80633; Cdp 840;
 Uk 88525; Uk 112166

=> s L5 and (zaprinast or dipyridamole or sildenafil)
 L7 9 L5 AND (ZAPRINAST OR DIPYRIDAMOLE OR SILDENAFIL)

=> display l7 all 9

L7 ANSWER 9 OF 9 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
 AN 96123943 EMBASE <<LOGINID::20070328>>
 DN 1996123943
 TI Drug-induced lung disease: Cardiovascular agents.
 AU Zitnik R.J.

CS Department of Internal Medicine, Yale University School of Medicine, New Haven, CT, United States

SO Journal of Respiratory Diseases, (1996) Vol. 17, No. 4, pp. 293-298. .
ISSN: 0194-259X CODEN: JRDIFQ

CY United States

DT Journal; General Review

FS 006 Internal Medicine
015 Chest Diseases, Thoracic Surgery and Tuberculosis
037 Drug Literature Index
038 Adverse Reactions Titles

LA English

SL English

ED Entered STN: 7 May 1996
Last Updated on STN: 7 May 1996

AB The main respiratory side effect of angiotensin converting enzyme inhibitors is a nonproductive cough. Drug withdrawal is usually indicated; however, the cough may resolve spontaneously or respond to inhaled cromolyn. Although β -blockers are generally avoided in patients with asthma or chronic obstructive pulmonary disease (COPD), esmolol is the agent of choice when β - blockade is required for unstable angina. For patients with asthma or COPD who have had a myocardial infarction, low doses of atenolol or metoprolol may be given, but discontinue therapy at any sign of worsening respiratory function. Hydralazine can cause the development of a lupus-like syndrome, while protamine has been associated with anaphylaxis and pulmonary hypertension.

CT Medical Descriptors:
 *angioneurotic edema: ET, etiology
 *angioneurotic edema: DI, diagnosis
 *angioneurotic edema: SI, side effect
 *angioneurotic edema: TH, therapy
 *coughing: DI, diagnosis
 *coughing: SI, side effect
 *coughing: TH, therapy
 *coughing: ET, etiology
 *drug induced disease: DI, diagnosis
 *drug induced disease: DT, drug therapy
 *drug induced disease: TH, therapy
 *drug induced disease: SI, side effect
 *drug induced disease: ET, etiology
 *pulmonary hypertension: DT, drug therapy
 *pulmonary hypertension: SI, side effect
 *pulmonary hypertension: ET, etiology
 *pulmonary hypertension: DI, diagnosis
 anaphylaxis: SI, side effect
 anaphylaxis: TH, therapy
 anaphylaxis: DI, diagnosis
 anaphylaxis: DT, drug therapy
 anaphylaxis: ET, etiology
 beta adrenergic receptor blocking
 clinical feature
 drug withdrawal
 human
 lung toxicity: DT, drug therapy
 lung toxicity: TH, therapy
 lung toxicity: SI, side effect
 lung toxicity: ET, etiology
 lung toxicity: DI, diagnosis
 review
 risk factor
 systemic lupus erythematosus: ET, etiology
 systemic lupus erythematosus: SI, side effect
 systemic lupus erythematosus: TH, therapy
 systemic lupus erythematosus: DT, drug therapy

systemic lupus erythematosus: DI, diagnosis

Drug Descriptors:

*cardiovascular agent: AE, adverse drug reaction

atenolol: AE, adverse drug reaction

beta adrenergic receptor blocking agent: AE, adverse drug reaction

calcium channel blocking agent

corticosteroid: DT, drug therapy

dipeptidyl carboxypeptidase inhibitor: AE, adverse drug reaction

dipyridamole: AE, adverse drug reaction

esmolol: AE, adverse drug reaction

histamine h1 receptor antagonist: DT, drug therapy

histamine h2 receptor antagonist: DT, drug therapy

hydralazine: AE, adverse drug reaction

isophane insulin: AE, adverse drug reaction

labetalol: AE, adverse drug reaction

metoprolol: AE, adverse drug reaction

pindolol: AE, adverse drug reaction

protamine: AE, adverse drug reaction

RN (atenolol) 29122-68-7; (dipyridamole) 58-32-2; (esmolol)
81147-92-4, 81161-17-3; (hydralazine) 304-20-1, 86-54-4; (isophane
insulin) 9004-17-5; (labetalol) 32780-64-6, 36894-69-6; (metoprolol)
37350-58-6; (pindolol) 13523-86-9, 21870-06-4; (protamine) 11061-43-1,
9007-31-2, 9012-00-4

CN Neutral protamine hagedorn insulin

=> FIL STNGUIDE

COST IN U.S. DOLLARS

SINCE FILE

TOTAL

ENTRY

SESSION

FULL ESTIMATED COST

16.81

35.56

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FILE CONTAINS CURRENT INFORMATION.

LAST RELOADED: Mar 23, 2007 (20070323/UP).

=> file biosis, medline, scisearch, embase

COST IN U.S. DOLLARS

SINCE FILE

TOTAL

ENTRY

SESSION

FULL ESTIMATED COST

1.38

36.94

FILE 'BIOSIS' ENTERED AT 17:28:28 ON 28 MAR 2007

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FILE 'SCISEARCH' ENTERED AT 17:28:28 ON 28 MAR 2007

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FILE 'EMBASE' ENTERED AT 17:28:28 ON 28 MAR 2007

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=> s sildenafil and arginine

L8 591 SILDENAFIL AND ARGININE

=> s sildenafil and L-arginine

L9 382 SILDENAFIL AND L-ARGININE

=> s sildenafil (s) L-arginine

L10 178 SILDENAFIL (S) L-ARGININE

=> display 110 all 178

L10 ANSWER 178 OF 178 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
AN 1999313603 EMBASE <<LOGINID::20070328>>
TI The effect of sildenafil on apomorphine-evoked increases in intracavernous pressure in the awake rat.
AU Andersson K.-E.; Gemalmaz H.; Waldeck K.; Chapman T.N.; Tuttle J.B.; Steers W.D.
CS W.D. Steers, Department of Urology, Box 422, Univ. of Virginia Hlth. Sci. Center, Charlottesville, VA 22908, United States
SO Journal of Urology, (1999) Vol. 161, No. 5, pp. 1707-1712. .
Refs: 30
ISSN: 0022-5347 CODEN: JOURAA
CY United States
DT Journal; Article
FS 028 Urology and Nephrology
030 Pharmacology
037 Drug Literature Index
LA English
SL English
ED Entered STN: 27 Sep 1999
Last Updated on STN: 27 Sep 1999
AB Purpose: The aim of this study was to develop a quantitative, awake animal model to investigate the effect of sildenafil on centrally-evoked erectile activity. Methods: Intracavernous pressures were recorded in awake, male Sprague Dawley rats after administration of apomorphine (100 or 250 µg./kg. subcutaneously). Sildenafil (100 µg./kg. intravenously) was then given 10 min. after a second dose of apomorphine. The time to first response, duration of response, and peak intracavernous pressure and area under the response, were measured before and after sildenafil. Results: Apomorphine produced rhythmic increases in intracavernous pressure. The pressure increase consisted of two components. The amplitude of the first, tonic response was 58 ± 3 mm. Hg, and a superimposed, burst-like increase in pressure elevated this further to 81 ± 6 mm. Hg. Bilateral transection of the pudendal nerves abolished the burstlike pressure changes; bilateral transection of the cavernous nerves prevented both responses. The duration of the apomorphine- induced increase in intracavernous pressure was significantly ($p = 0.003$) prolonged by sildenafil (100 µg./kg.) from 37 ± 4 to 62 ± 11 s ($n = 6$). The overall intracavernous pressure response to apomorphine (100 µg./kg.), measured as the area under the curve, was significantly ($p = 0.003$) increased by sildenafil (100 µg./kg.) from 67 ± 8 to 142 ± 31 units ($n = 6$), N- nitro-L-arginine methyl ester (40 mg./kg. intravenously) prevented the apomorphine-induced responses. Conclusions: Monitoring intracavernous pressures in the awake rat represents a simple model to evaluate the effect of drugs on erectile function. Using this model we have shown that apomorphine elicits a rise in intracavernous pressure that can be prolonged by sildenafil. These results suggest that there may be a role for the combination of apomorphine and sildenafil in the management of erectile dysfunction.
CT Medical Descriptors:
*drug effect
*erectile dysfunction: DT, drug therapy
pressure measurement
area under the curve
impotence
pudendal nerve
nerve transection
drug potentiation
nonhuman
male
rat
animal experiment

animal model
controlled study
adolescent
intravenous drug administration
article
priority journal
Drug Descriptors:
*apomorphine: CB, drug combination
*apomorphine: IT, drug interaction
*apomorphine: DT, drug therapy
*sildenafil: CB, drug combination
*sildenafil: IT, drug interaction
*sildenafil: DT, drug therapy

RN (apomorphine) 314-19-2, 58-00-4; (sildenafil) 139755-83-2

=> s sildenafil and L-arginine

L11 382 SILDENAFIL AND L-ARGININE

=> display l11 382 all

L11 ANSWER 382 OF 382 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN

AN 1999114920 EMBASE <<LOGINID::20070328>>

TI Effects of castration and androgen replacement on erectile function in a rabbit model.

AU Traish A.M.; Park K.; Dhir V.; Kim N.N.; Moreland R.B.; Goldstein I.

CS Dr. A.M. Traish, Biochemistry and Urology, Department of Urology, Boston University School of Medicine, 700 Albany Street, Boston, MA 02118, United States. atraish@bu.edu

SO Endocrinology, (1999) Vol. 140, No. 4, pp. 1861-1868. .

Refs: 38

ISSN: 0013-7227 CODEN: ENDOAO

CY United States

DT Journal; Article

FS 003 Endocrinology

LA English

SL English

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AB We investigated, in a rabbit model, the effects of castration and testosterone replacement on: 1) the hemodynamics of the corpus cavernosum; 2) α -1 adrenergic receptor protein expression; 3) neural NO synthase protein expression and activity; 4) phosphodiesterase type 5 activity; and 5) trabecular smooth muscle/connective tissue balance. One week after bilateral orchiectomy, animals were treated for 7 days with vehicle alone, testosterone, or estradiol. Intact control animals received vehicle only. Systemic arterial blood and intracavernosal pressures (ICP) were measured in each animal before and after electrical stimulation of the cavernosal nerve, α 1-adrenergic receptor protein expression was determined by ligand binding studies. NO synthase expression and activity were determined by Western blot analyses and conversion of L-arginine to citrulline, respectively. Phosphodiesterase type 5 activity was determined by hydrolysis of guanosine 3',5'-cyclic monophosphate (cGMP) in tissue extracts in the absence or presence of 100 nM sildenafil. Smooth muscle content was assessed by Masson's trichrome staining and computer-assisted histomorphometry. Castration significantly reduced ICP, but it did not alter systemic arterial blood pressure during stimulation of the cavernosal nerve. Testosterone, but not estradiol, treatment prevented the effects of castration and restored ICP to values similar to those obtained in intact animals. Castration reduced expression of α 1-adrenergic receptor, and this reduction was prevented or reversed by testosterone replacement. Neural NO synthase protein expression and total activity were not altered significantly by

castration or after testosterone replacement. However, phosphodiesterase type 5 activity increased in castrated animals treated with testosterone. Castration significantly reduced trabecular smooth muscle content, and this reduction was restored by testosterone (but not estradiol) treatment. The results of this study demonstrate that androgen deprivation alters the functional responses and structure of erectile tissue.

CT Medical Descriptors:

*castration

*penis erection

rabbit

hormone substitution

corpus cavernosum

protein expression

structure activity relation

nonhuman

animal model

article

priority journal

Drug Descriptors:

*androgen

nitric oxide synthase

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